

## ESTIMATION OF AST,ALT AND COMPLETE LIPID PROFILE IN HUMAN INFECTED BY *ENTAMOEBIA HISTOLYTICA* IN AL-NAJAF GOVERNORATE, IRAQ<sup>+</sup>

Maysoon Khudar Al-Hadrawy \*

### Abstract :

The study was conducted 60 out patients and 30 healthy people to determine the effect of infection with *Entamoeba histolytica* on levels of AST ,ALT, Total cholesterol, TG, HDL cholesterol, LDL cholesterol and VLDL cholesterol in human infected with *Entamoeba histolytica* compared with healthy group . Who have visited Al-Sadder medical city, Al-Hakeem Hospital ,AL-Zahraa Hospital , AL-Manaithra Hospital in Al-Najaf governorate during the period from October 2013 till April 2014. The results showed that here were a significant increase (P<0.05) in AST ,ALT. while there was a significant decrease in cholesterol, TG,LDL, HDL, and VLDL in *Entamoeba histolytica* infected patients compared with control group.

Key words : Low density Lipoprotein cholesterol, High density Lipoprotein cholesterol, very Low density Lipoprotein cholesterol, *Entamoeba histolytica*

تقييم ناقل الأسبارتات وناقل الألائين وصورة الدهون لدى المصابين بطفيلي أميبا الزحار في محافظة النجف ، العراق

ميسون خضير عبد العباس

### المستخلص :

صُممت هذه الدراسة لتحديد تأثير الإصابة بطفيلي *Entamoeba histolytica* على مستوى ناقله الاسبارتات AST وناقله الألائين ALT وصورة الدهون ( الكوليستيرول ,الكليسرايد الثلاثي, الدهون البروتينية العالية الكثافة , الدهون البروتينية الواطئة الكثافة , الدهون البروتينية الواطئة الكثافة جداً ), حيث أشتملت الدراسة الحالية على ( 60 ) حالة إصابة بالطفيلي و( 30 ) حالة من أشخاص غير مصابين وافدين الى مدينة الصدر الطبية ومستشفى الحكيم ومستشفى المنارة ومستشفى الزهراء في محافظة النجف الأشرف للمدة من شهر كانون الثاني 2013 ولغاية شهر نيسان 2014 . أظهرت الدراسة الحالية حصول زيادة معنوية في كل من الـAST و الـALT ,وقلة في مستوى الكوليستيرول والكليسرايد الثلاثي والدهون البروتينية الواطئة الكثافة والواطئة الكثافة جدا و مستوى الدهون البروتينية العالية الكثافة مقارنة بمجاميع السيطرة .

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\* Lecturer / Technical institute / kufa / AL-Furat AL-Oust Technical University

## **Introduction :**

The parasitic infections caused by intestinal helminthes and protozoan parasites are among the most widespread human infections in developing countries [1]. In developed countries, protozoan parasites more commonly cause gastrointestinal infections compared to helminthes. The most common intestinal protozoan parasites is *Entamoeba histolytica* caused Amoebiasis [2] .

Amoebiasis is an infection caused by *Entamoeba histolytica* with or without symptoms [3] ,it is worldwide in distribution and continues to be an important public health problem. This parasite is transmitted to humans by way of contaminated water and food. The diagnosis of amoebiasis is often difficult and time consuming. The main purpose of detection and differentiation of *E. histolytica* species in stool samples is the detection of the causative agent of amoebic dysentery [4].

*Entamoeba histolytica* is a unicellular protozoan parasite that causes about 50 million infections with a death rate of over 100 000 universal yearly [5]. The amoebic infection is the third most widespread cause of death among parasitic diseases, after malaria and schistosomiasis [6].

The occurrence of parasitic infections varies in different parts of the world. The prevalence of *Entamoeba histolytica* range from 5% to 81% , geographical conditions and poor nutritional and socioeconomic status contribute to making Iraq a favourable area for parasitic infections [7].

*Entamoeba histolytica* has a simple life cycle, in which the transmission is via the faecal-oral route. Infection occurs through ingestion of infective cysts (size 8-20  $\mu\text{m}$ ) or invasion of motile trophozoites (size 20-40  $\mu\text{m}$ ) [8]. Most infections are asymptomatic, but *E.histolytica* can invade the gut wall, causing severe ulceration and amoebic dysentery characterized by bloody stools (9,10). If the parasites gain access to damaged blood vessels, they may be carried extra intestinal sites anywhere in the body, the most important of which is the liver, where the amoebae cause hepatic amoebiasis (liver abscess) [11].

The aim of this study is measure effect of infection by *E. histolytica* on levels of AST ,ALT and complete lipid profile Tri- glyceride (TG), Total cholesterol, High Density Lipoprotein (HDL) ,Low Density Lipoprotein (LDL),and Very Low Density Lipoprotein (VLDL) in serum of patients in Al-Najaf governorate and compared with control groups.

## **Materials and Methods :**

### **Blood collection :**

The study comprised 60 infected patients with *E. histolytica* selected among 306 patients suffering abdominal pain in Al Sadar Education hospital,AL-Zahraa hospital,AL- Manaithara hospital and Al-Hkeem hospital at the period from October 2013 to April 2014 in Al-Najaf governorate. The patients were diagnosed on the base clinical finding and microscopically positive stool examination for *E. histolytica* .Five ml from each of blood samples was drawn from each patients and control group in sterile tubes and remains for 30 minutes at room temperature. The samples were centrifugation at 3000 rpm for 5 minutes to separate the serum and collected in another sterile tubes, each sample of serum kept at -20C°. Measurement of lipid:

**\* Estimation of Total Serum Cholesterol:**

Measurement of serum Total cholesterol by enzymatic and colorimetric method Where cholesterol esterat lysis to cholesterol and fatty acid by cholesterol esterase[12].

**\* Estimation of SerumTri- glycerid (TG):**

Measurement of TG in the serum by enzymatic and colorimetric method ,the Tri-glycerids in serum lysis enzymatically to Glycerol Phosphate and fatty acid by Lipase [13].

**\* Estimation of Serum High Density Lipoprotein (HDL):**

Measurement of ( HDL) in the serum by sedimentation Lipoproteins found with HDL ,using phosphotungstic acid solution with found  $Mg^{+}$  [14 ]. The very low density Lipoprotein concentration was calculated by using the following formula [15]

$$VLDL. Cholesterol( mg/100 ml) = TG /5$$

The Low Density Lipoprotein Concentration was calculated by using the following formula Low density lipoprotein =Serum cholesterol - (VLDL+HDL) . [16]

**\* Estimation of AST:**

Was measured enzyme activity Glutamate oxaloacetate transaminase (AST) using a kit supplied by (Randox) [ 17], which is based on the ability of the enzyme to convert Aspartic acid to Oxaloacetic acid, which automatically turns to Pyrovic acid and then the interaction of the latter with a compound 2,4-dinitrophenyl hydrazine (DNPH) to form a complex with a brown color - reddish measured absorbance him at a wavelength (546) nm.

**\* Estimation of ALT:**

The activity of Glutamate pyruvate transaminase (ALT) was measured by using Randox kit [17].

**Statistical analysis:**

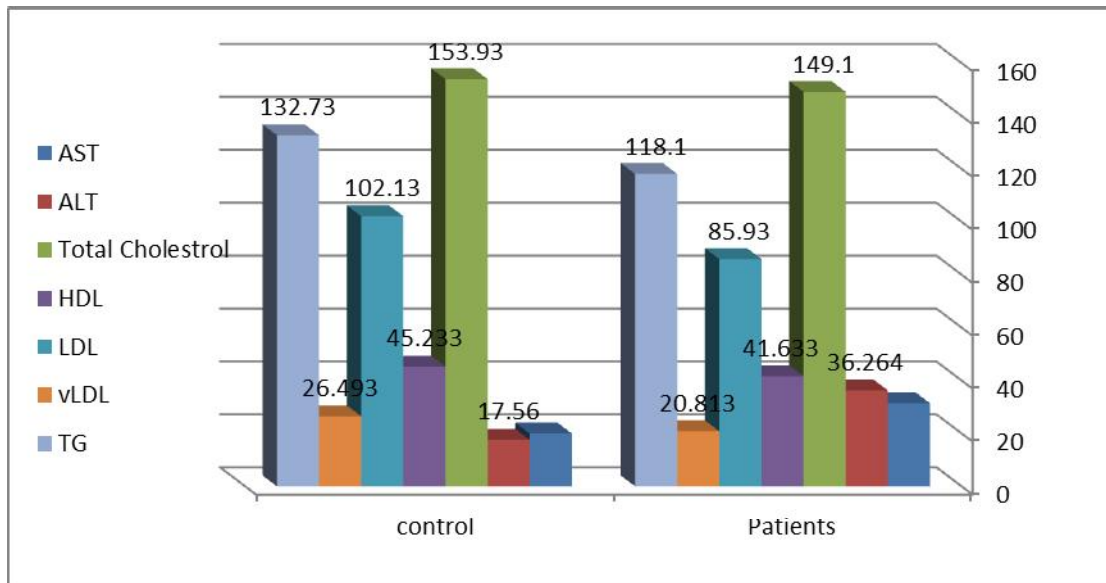
Data were analyzed using the software packages Graphpad prism for Windows (5.04, Graphpad software Inc. USA), Data are presented as the mean  $\pm$  standard error (SE). The comparison between the patients and healthy groups were analyzed by one-way ANOVA. A p-value < 0.005 was considered significant.

**Results :**

Table (1) and Figure (1) Comparison of the studied parameters between patients infected by *E. histolytica* and control group

Table (1)

Mean of parameters $\pm$ SD	patients	control	P value
AST(Mg/dl)	31.4	19.968	P<0.05
ALT(Mg/dl)	36.264	17.56	P<0.05
Total Cholestrol(Mg/dl)	149.1	153.93	P<0.05
HDL(Mg/dl)	41.633	45.233	P<0.05
LDL(Mg/dl)	85.93	102.13	P<0.05
vLDL(Mg/dl)	20.813	26.493	P<0.05
TG(Mg/dl)	118.1	132.73	P<0.05



\*Significant difference between control group and patients (P<0.05)

Figure (1): comparison of studied parameters between patients and Healthy group

## Discussion :

The results revealed a significantly lower lipid profile was apparent in *Entamoeba histolytica* patients compared to the control group. This results due to the pathogenicity of this parasite dependent on the relationship between serum lipid profile concentration and adhesion of parasite on epithelial cell [18].

Changes of protein fractions AST, AIT varied according to the qualitative difference in the intensity of inflammation by strains of *E. histolytica*. In *E. histolytica* infection, changes in cholesterol and lipid level show a greater association with active infection those results in intestinal amoebiasis. In intestinal amoebiasis it is probable that cholesterol absorption from the intestine is reduced, while in liver abscess cases it may be depleted from serum [19]. That cholesterol/lipids may have a role in the pathogenesis of *E. histolytica*, and is corroborated by studies that demonstrate that *E. histolytica* becomes more virulent in the presence of cholesterol. [20,21].and be due to the parasites utilise cholesterol for their growth in infected individuals [22], Cholesterol is thought to act as an irritant on mucous membrane and thus helps the amoebae to colonies the injured site, enhancing the parasite's virulence, changes in cholesterol and lipid level show a greater association with active infection that results in intestinal amoebiasis. In intestinal amoebiasis it is probable that cholesterol absorption from

the intestine is reduced, [23]. Trophozoite in the intestine converts to cysts in response to the presence of host factors, such as bacterial flora, that might use up the cholesterol needed for encystations[24 ] .

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